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Child Abuse and Dentistry:

A Study of Knowledge and Attitudes Among Dentists in Victoria, Australia

Abstract of a research project conducted by fifth year dental students at The School of Dental Science, The University of Melbourne, 1997-8.
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In recent years, the community has become increasingly aware of the problem of child abuse in society. Child abuse may be defined as an act by parents or care givers which endangers a child's or young person's physical or emotional health or development.

Four types of child abuse are recognised: physical abuse, sexual abuse, emotional abuse and neglect. Section 64(1) of the Children and Young Persons Act 1989 (Victoria) allows any person to notify any instance of suspected or known child abuse. In 1993, this legislation was amended to require mandatory reporting of suspected cases of child abuse by certain professionals when, in the course of practicing their profession, they form a belief, on reasonable grounds, that child is in need of protection from abuse. Individuals mandated to report suspected child abuse are police, nurses, doctors, psychologists, teachers, child care workers, owners and operators of child care centres, parole and probation officers, and social and youth workers. Dentists are not mandated to report child abuse in Victoria, but they are mandated in South Australia and the Australian Capital Territory.

Very little has been written in the dental literature regarding the role of the Australian dental profession in the recognition and reporting of child abuse. The aims of the present study of dentists in Victoria were two fold: (1) to assess the current status of their knowledge and attitudes about child abuse, and (2) to increase their awareness of child abuse. Of the 530 dentists approached, only 496 could be contacted and of these, 347 participated (88%). The sample contained members of the ADAVB (n=141), new graduates group (n=59), ASE (n=102) and ANZSPD (n=45). The pre-tested survey instrument (a questionnaire administered either in person or by telephone) contained 16 open-ended or multiple choice questions which surveyed practice characteristics, knowledge of child abuse, attitudes towards child abuse, previous experience of the problem, and their management of child abuse. Over 80% of responding dentists had graduated since 1970 and children were seen regularly in most practices studied.

Overall, this study of knowledge and attitudes of dentists about child abuse demonstrated a poor understanding of the problem, despite a very high level of interest as demonstrated by the high participation rate and a strong desire for further information by the participants. Overall, most were well able to define child abuse in terms of physical and emotional aspects (88-99%), and provide psychosocial and medical signs of child abuse (77-99%). Most respondents (75%) could identify the health department, community services, police, social worker or the

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President's Report

Every generation of dental practitioners convinces themselves that they have to cope with more change than the previous one. I am sure we feel likewise at the present time. At all levels of Government there are numerous changes which could have significant effects on dental practice. We are all familiar with the federal governments influence on both public and private dentistry. In the public sector the cancellation of the additional funding for the Commonwealth Dental Scheme was a significant blow for dentistry. This Scheme was aimed at the dentate, needy, adult who had for so long missed out on obtaining dental care. However the flow on affect of the Scheme to dentistry in general, was very important.

In the private sector the federal government's push to free up competition in all spheres of Australian life and the involvement of some State Governments in advancing this cause, may yet be one of the most serious challenges for all of us, particularly the proposed changes to Victorian Dental Act. There are passionate arguments from dentists on both sides of fence. However I feel very strongly that when a consumer seeks dental care they have a right to have confidence in the registered qualifications of the dentist. Any attempts to alter these accepted community standards needs to be very carefully considered.

The differing beliefs in the importance of government regulation in our social systems seems to be showing up as the fundamental difference between our two main political parties. This will no doubt become a very important point for the voting public in choosing which way they would like our society to move. For us as dentists, passionately interested in the dental health and general well being of children we must increase our role as advocates of high quality child health and where this is threatened present cogent arguments, to governments, oppositions and the bureaucracy, otherwise, sadly, we will be left out of the health equation.

This years dental health week will be held from he 2nd - 8th of August and the theme will be 'Sports Dentistry'. Could I urge all of you to become actively involved in this promotion, and use the material that will be provided by the ADA. This is a classic area where our advocacy must take us not only to our own patient pool & the wider community but also to all levels of government, where standards are set for such things as mouth guard materials, safety requirements for sporting clubs and promotion of child safety.

As a result of the Council Meeting in Perth during the ADA congress it was decided to hold a special conference of the ANZSPD in Adelaide in July of next year. The Australian Academy of Paediatric Dentistry have expressed strong interest in co-hosting this meeting. The aim of the meeting will be to spend two days, developing 'standards of care' for our organisations. I would like a facilitator used to keep the work on track and make sure our time is productive. Mid July was settled upon, so as not to clash with the Trauma Conference in Melbourne. I will be writing to all state secretaries in the near future outlining the details of the meeting.

Finally, let me congratulate John Winters on producing an excellent synopses first time around. May he continue in the job for many years.

Richard Widmer
President, ANZSPD

Women's And Children's Health Care Network
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Senior Registrar in Paediatric Dentistry

Department of Dentistry

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Further information is available from:

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Periodontal Diseases in Children and Adolescents

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Periodontal diseases affecting children and adolescents are characterised by onset prior to age 35 and often involve a rapid rate of periodontal destruction. Periodontal diseases are not only infective in nature but are also mediated by host parasite interactions. On occasion, periodontal diseases reflect hidden systemic disorders which have not been diagnosed.

According to the 1996 position paper of the Committee on Research, Science and Therapy of The American Academy of Periodontology (AAP), clinically distinct periodontal infections that can affect young individuals include: 1) chronic gingivitis; 2) early onset periodontitis; 3) necrotizing ulcerative gingivitis/periodontitis; and 4) periodontitis associated with systemic diseases.[2] Generalised prepubertal periodontitis (PP) is now included in the latter group i.e. periodontitis associated with systemic diseases. Of interest, the AAP classification does not include children with prepubertal periodontitis who are otherwise healthy. This paper will review the normal periodontium in children (primary dentition) and then address specific periodontal diseases found in children and adolescents.

The normal periodontium of the primary dentition

Gingival tissues of young children differ from those of adults in their clinical appearance and resistance to the development of gingivitis and periodontitis. The gingiva appears redder (less pink) and less dense than in adults because of increased vascularity, and has a thinner, less keratinized and more translucent epithelium. It is also more flaccid, retractable and resilient in consistency because of lower connective tissue density. The gingival surface appears smooth or slightly stippled. The marginal gingiva is round and thick because of the cervical bulge of the primary dentition and the underlying constriction at the cementoenamel junction. The average sulcular pocket depth is 2 mm. The width of attached gingiva is comparatively greater than that found in adults. The maxillary attached gingiva is wider than mandibular attached gingiva. The interdental col found in the primary dentition without spacing between the teeth has nonkeratinized epithelium and is vulnerable to bacterial growth and secondary tissue invasion. On the other hand, the interdental saddle, a well keratinized surface, is found in the primary dentition with generalised spacing between the teeth. This area is less vulnerable to the development and progression of the inflammatory process.

The interdental cleft and the retrocuspid papilla are two unique anatomical characteristics found in the attached gingiva in children. Interdental clefts are normal anatomical features found in the interradicular zone underlying most interdental saddle areas. The retrocuspid papilla is found approximately 1 mm below the free gingival groove in the attached gingiva lingual to the mandibular primary canine. This feature appears to have no clinical importance but may be mistaken for a sinus tract.

In comparison with the permanent dentition, the alveolar bone surrounding the primary teeth is less calcified, more vascular, and has fewer but thicker trabeculae, larger marrow spaces and flatter interdental crests. These features indicate the dynamic and transitional metabolic state of young and maturing bone. Children also have a wide periodontal membrane but thinner lamina dura.[3-5]

Chronic gingivitis

Chronic gingivitis is a non specific infection associated with bacterial plaque and is the most common form of periodontal disease in children.[3,4] It is characterised by painless, localised or generalised gingival inflammation usually limited to the marginal and papillary gingiva without detectable loss of bone or clinical attachment.[2] Spontaneous haemorrhage is evident only in more severe cases. The local factors that contribute to the development of gingivitis include plaque and calculus accumulation, traumatic injury from tooth exfoliation or eruption, carious teeth, food impaction, malocclusion, orthodontic appliances, mouth breathing, and faulty restorations with marginal overhangs or improper extension of stainless steel crowns[3]. Children experience gingivitis to varying degrees, however the gingivitis is usually less severe than gingivitis in an adult with similar levels of plaque. Gingivitis in children appears to be reversible and rarely progresses to periodontitis.[4,5]. Gingivitis in children usually responds to a thorough professional removal of bacterial deposits and improved daily oral hygiene practices. With the resumption of effective oral hygiene, gingivitis will be eliminated within seven days.[3]

Early-onset periodontitis (EOP)

Early-onset periodontitis (EOP) describes a group of severe periodontal diseases affecting young individuals who are otherwise healthy. The age of onset is near puberty.[2] There are two types of EOP usually described: localised juvenile periodontitis (LJP) and generalised juvenile periodontitis (GJP).[2,6]

Localised juvenile periodontitis (LJP)

Localised juvenile periodontitis is a localised form of EOP found in teenagers and young adults without clinical evidence of systemic disease. It is characterised by rapid vertical or angular alveolar bone resorption, mainly affecting first permanent molars and incisors.[1,7-9] There is a slight gingival inflammation accompanied with small amount of supra or subgingival plaque and calculus.[2] The estimated prevalence varies from 0.1% to 15%.[1] The etiological factors are not known and there is not a strong association between the disease and the amount of plaque or calculus. Localised juvenile periodontitis is most often associated with the bacterium *Actinobacillus actinomycetemcomitans* (*Aa*).

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However, other putative etiological bacteria include *Bacteriodes* and *Eubacterium sp.*, *Prevotella intermedia, Capnocytophaga ochracea, Eubacterium nodotum* and *Peptostreptococcus micros.*[2,7,10,11] *Actinobacillus actinomycetemcomitans* is the most important actinobacillus involved in periodontal disease. It is a gram-negative, non-motile facultatively anaerobic rod. The species contains five serotypes (a to e). *Actinobacillus actinomycetemcomitans* serotype b is the most common serotype in LJP.[23] Serotype a is associated with adult periodontitis and serotype c is associated with extraoral infections. *Actinobacillus actinomycetemcomitans* has a number of factors and mechanisms that serve as virulence determinants. These virulent factors include bacteriocin, chemotaxis inhibitor, immunosuppressive factors, leukotoxin, invasins, Fc binding proteins, endotoxin, cytotoxin, collagenase and adhesins.[24]

Generalised juvenile periodontitis (GJP)

Generalised juvenile periodontitis is a generalised form of EOP found in older juveniles and young adults, and may involve the entire periodontium.[2] Alternative terms for GJP have been used such as severe periodontitis, severe generalised periodontitis and rapidly progressive periodontitis.[2,12] Unlike LJP, patients with GJP exhibit severe generalised gingival inflammation, bone resorption, gingival bleeding and tooth mobility.[8,9] Also, heavy supragingival and subgingival plaque and calculus accumulations occur.[8] Possible pathologic bacteria found in subgingival sites include black-pigmented *Bacteroides* and non specific black-pigmented anaerobic rods.[7,9] *Prophyromonas gingivalis* is strongly associated with this disease[10]. Neutrophils in GJP patients usually exhibit a deficiency in neutrophil chemotactic migration[8,9]. There may be an association between LJP and GJP since many studies have reported families manifesting EOP[8,9]. If left untreated, a delayed diagnosis of LJP may lead to progression of the disease and the development of GJP[7]. However, it has been suggested that LJP and GJP may be different disease entities and that the conversion from LJP to GJP is unlikely.[12]

Treatment of LJP and GJP depends on early diagnosis, direct elimination of pathologic bacteria, and providing an environment for healing[2]. Localised juvenile periodontitis usually responds well to mechanical therapy consisting of root planing and scaling, or root planing and scaling with open flap curettage[12]. The recommended antibiotics are tetracycline alone or and in combination with metronidazole[2]. Tetracycline should not be used in children under 8 years old due to the possibility of incorporation into mineralising teeth. The usual dose is 250-500 mg/dose every 6 hours.[20] The dose for metronidazole is 7.5 mg/kg/dose every 8 hours (adult dose is 200-400 mg/dose 8 hourly)[20]. Generalised juvenile periodontitis does not respond well to the same treatment as LJP and may require additional approaches such as root planing with flap debridement followed by frequent professional cleaning and scaling.[12]

Necrotizing ulcerative gingivitis/ periodontitis (NUG/P)

Necrotizing ulcerative gingivitis/periodontitis (NUG/P) is more common in young adults than children[13]. The significant findings used in the diagnosis of NUG/P are the presence of interproximal necrosis or punched-out interdental papillae, a pseudomembranous necrotic covering of the marginal tissue and rapid onset of gingival pain. The clinical manifestations of the disease include inflamed, painful and bleeding gingivae, poor oral hygiene, poor appetite, fever, general malaise and halitosis[13].

Currently, no predictably successful treatment other than tooth extraction is known. Impeccable oral hygiene, including the use of chemical plaque control agents and frequent professional tooth cleaning , is a recommended treatment.

The aetiology is of bacterial origin. Microorganisms believed to be responsible for the disease include *Borrelia vincentii*, spirochetes and *Prevotella intermedia*[3,13]. Malnutrition, viral infection or systemic illness, emotional stress and lack of sleep may be predisposing factors.[2,3] Depression in polymorphonuclear leukocyte phagocytosis of certain bacteria (*C. rectus, P. intermedia*) has been reported.[19] The lesions of ANUG/P respond dramatically, within 48 hours, to oral cleansing and mouth rinsing with diluted hydrogen peroxide or other oxidising agents. Antibiotic therapy is indicated when a patient exhibiting acutely and extensively inflamed gingiva is first seen.[18]

Acute necrotizing gingivitis (ANUG), an acute form limited to gingival tissues, should be differentiated from acute herpetic gingivostomatitis[13]. Therapeutic trial cleaning will bring about a favourable response in ANUG but will not be effective for acute herpetic gingivostomatitis. Acute necrotizing gingivitis, but not primary herpetic gingivostomatitis, will respond to antibiotic therapy. Acute necrotizing gingivitis/periodontitis is characterised by the clinical appearance described above involving the destruction of tooth supporting structures and always develops from pre-existing ANUG[14]. Acute necrotizing gingivitis/periodontitis has assumed greater importance in recent years because it is frequently observed as one of the oral symptoms of HIV-seropositive and AIDS patients.[14]

Periodontal disease associated with systemic disease

Defects of neutrophil and immune cell function are factors associated with some systemic diseases where there is an increased susceptibility to periodontal disease as well as to other infections[2]. Diseases in children, which manifest periodontal destruction, include leukocyte adhesion deficiency, Papillon Lefevre syndrome, hypophosphatasia, cyclic neutropaenia, leukemia and Type I diabetes mellitus.

Periodontitis associated with leukocyte adhesion deficiency (LAD)

Leukocyte adhesion deficiency (LAD) is a rare disease. Periodontal disease associated with LAD was termed generalised prepubertal periodontitis (GPP) by Page *et al.* in 1983[15]. The underlying cause of GPP is a genetic abnormality (autosomal dominant) of leukocyte receptors (CD11) on the surface of the phagocytic blood cells which leads to a defect in phagocytosis. Consequently, affected children are highly susceptible to infections including periodontal infection, chronic or recurrent infections of the upper respiratory tract, middle ear (otitis media), and skin infections. Clinical manifestations of periodontal disease include acute inflammation with clefting and recession of gingiva, rapid destruction of alveolar bone and, in some cases, root resorption.

Currently, no predictably successful treatment other than tooth extraction is known. Impeccable oral hygiene, including the use of chemical plaque control agents and frequent professional tooth cleaning , is a recommended treatment.[5]

The localised form of prepubertal periodontitis (LPP), on the other hand, is more frequent than the generalised form, but it too is relatively rare. Localised prepubertal periodontitis patients seem to be

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apparently healthy without a history of recurrent infection. Page found that although the CD11 family adherence receptors expressed by phagocytic cells in these children are normal, these cells have other abnormalities in functions such as chemotaxis5. The disease begins soon after eruption of primary teeth and if left untreated may persist into the mixed dentition. The disease is manifested by rapid alveolar bone loss detectable on radiographs and by periodontal pocket formation. Gingival tissues may be free of clinical signs of inflammation. Putative etiological factors in the development of periodontitis in otherwise apparently healthy children before puberty include pathogenic bacteria such as *Actinobacillus actinomycetemcomitans, Prevotella intermedia, Eikenella corrodens* and *Capnocytophaga sputigena*[2]. Localised prepubertal periodontitis responds very well to thorough root planing combined with systemic antibiotics (tetracycline and metronidazole), combined with excellent oral hygiene.[5]

Generalised prepubertal periodontitis (or LAD) and LPP are considered traditionally as prepubertal periodontltis, affecting periodontal tissues in children before puberty, mostly in the primary dentition. Early diagnosis is very important and this should be based on clinical and radiographic manifestations, and history of underlying systemic disease.

Systemic diseases that should be differentiated from LPP include Papillon Lefevre syndrome, hypophosphatasia, neutropaenia, histiocytosis, Chediak-Higashi syndrome, leukemia, and juvenile diabetes mellitus.

Other systemic diseases manifested periodontal destruction

Papillon Lefevre syndrome (LPS) is characterised by hyperkeratosis of the palms and soles, severe destruction of alveolar bone and early loss of primary and permanent teeth. Virulent gram-negative anaerobic pathogens such as *Actinobacillus actinomycetemcomitans* are responsible for the periodontal destruction. The recommended treatment of PLS is the elimination of the source of infection by a removal of all primary teeth before the permanent dentition has erupted. This will prevent further infection to permanent teeth[16]. A recent case report showed that frequent recalls and professional cleaning in combination with periodontal irrigation with antiseptic agent, in coupled with special tooth brushing technique named "tooth pick method" has successfully preserved permanent teeth in a Japanese patient with Papillon-Lefevre syndrome.[25]

In hypophosphatasia, the patient exhibits premature loss of anterior primary teeth without root resorption due to partial or complete lack of cementum formation, and bone deformities especially of the legs. The patient also exhibits calcification failure of the calvarium and growth retardation. The presence of phosphoethanolamine in the urine and reduced serum alkaline phosphatase activity are diagnostic features.[22]

Neutropaenia is a quantitative neutrophil disorder characterised by a neutrophil cell count less than 1.8 x 109 cells/litre[17]. Cyclic neutropaenia is characterised by the disappearance of neutrophils from the blood and bone marrow, at regular intervals of approximately 3 weeks or 25-28 days. Serial studies are needed to confirm the diagnosis of cyclic neutropaenia. Neutropaenic patients experience severe periodontal infection manifested by marked gingival inflammation, oedema, gingival recession and loss of alveolar bone.[4,21]

Chronic gingivitis, early onset periodontitis, necrotizing ulcerative gingivitis/periodontitis and prepubertal periodontitis are specific periodontal diseases occurring in children and adolescents.

Treatment of periodontitis in children with neutropaenia begins with aggressive measures to prevent periodontal bone loss. Strictly controlled oral hygiene is very important. Poor or uncontrolled oral hygiene will result in worsening the oral status.[21] Early referral and the identification of the disease by physicians is required.

Acute leukemia is a malignancy of hematopoietic tissues characterised by acute gingival enlargement, gingival ulceration and spontaneous gingival bleeding. It is diagnosed by the presence of a disseminated proliferation of immature leukocytic cells in the bone marrow and body organs including gingiva. The periodontal destruction and tooth migration are a result of leukocytic infiltration.

The clinical manifestations of histiocytosis X range from solitary or multiple bone lesions to disseminated visceral, skin and bone lesions. The skull, mandible, ribs, vertebrae, and long bones are frequently involved. Lesions in the oral cavity may be the first indication of the disease, presenting as pain, gingival swelling, or loosening of the teeth. Oral lesions characteristically occur in all four quadrants. This lesion extends forward to the canines, but rarely involves the incisors. Radiographically, teeth appear to be "floating in space". Other symptoms include malaise, irritability, ano-vuval and postauricular rash, and diabetes insipidus. Biopsy of the lesion, positive immunofluorescece to S100 protein and CD1 antigen, and the presence of Birbeck's granules on histopathological examination provide the definitive diagnosis. Conservative excision and curettage of oral lesions and fluorescence extraction of involved teeth is required to control oral lesions. Advanced lesions require surgical excision, curettage and extraction combined with chemotherapy.[27]

Chediak-Higashi syndrome is characterised by occulocutaneous albinism with photophobia and nystagmus, frequent pyogenic infections and intermittent febrile episodes. Severe gingival inflammation appears to be a common finding, with excessive and early periodontal destruction resulting in premature loss of teeth. A diagnostic feature of this syndrome is the haematological finding of giant, abnormal granules in the peripheral circulating leukocytes.[18] Successful treatment is difficult due to the qualitative neutrophil defect, abnormal B-cell and T-cell function, and thrombocytopaenia. As with leukocyte adhesion deficiency, granulocyte transfusion and bone marrow transplantation may help by improving the resistance to infection.[26] Children die by ten years of age because of overwhelming sepsis.[26]

Periodontal disease is the most consistent oral finding in patients with poorly controlled juvenile diabetes mellitus because these children exhibit altered neutrophil chemotaxis.[4] Polydipsia, polyuria, weight loss with polyphagia, and recurrent infections including candidiasis are clinical symptoms.[17] Diagnosis of diabetes is made by the detection of fasting blood glucose level above 120 mg/dl, abnormal glucose tolerance test and elevated glycosylated haemoglobulin test values. Successful treatment includes excellent oral hygiene and control of blood glucose levels.[4] Treatment of periodontal disease in this patient should concentrate on excellent oral hygiene practice using a chlohexidine gluconate brush-on gel and/or chlohexidine mouth rinse and prophylaxis. Antibiotic therapy such as tetracycline and metronidazole should be initiated for the

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periodontal condition.[27]

Discussion

Chronic gingivitis, early onset periodontitis, necrotizing ulcerative gingivitis/ periodontitis and prepubertal periodontitis are specific periodontal diseases occurring in children and adolescents. Chronic gingivitis is the most common and is directly associated with plaque and calculus deposition. Therefore, it responds very well to local debridement and excellent oral hygiene. Early-onset periodontitis and prepubertal periodontitis are characterised by rapidly progressive periodontal destruction with the generalised forms affecting most of the teeth and exhibiting more severe periodontal destruction than the localised forms. Prepubertal periodontitis is found in prepubertal individuals in the primary dentition. Only generalised prepubertal periodontitis is associated with the systemic disease, leukocyte adhesion deficiency; localised prepubertal periodontitis, localised juvenile periodontitis and generalised juvenile periodontitis affect otherwise healthy individuals. The elimination of pathogenic bacteria and the maintenance of oral hygiene are sufficient to control the progression of the localised prepubertal periodontitis, localised juvenile periodontitis and generalised juvenile periodontitis in healthy patients. On the other hand, a poorer prognosis is expected in patients with systemic disease and extraction of severely affected teeth may be necessary.

When periodontal disease is detected in the primary dentition, a thorough examination including physical, oral and radiographic information, and blood chemistry should be performed. It is recommended that the following minimal tests should be performed in cases designated as localised prepubertal periodontitis. Complete blood count, white blood cell count, differential and cell morphology, serum alkaline phosphatase level, fasting glucose level, examination of palms and soles for hyperkeratosis and gingival biopsy including curetted tissue from the bony lesion[19]. Patients should be referred for medical investigation if systemic diseases are suspected. Prognosis of the disease depends on pathogenesis and the compliance of patients in maintaining excellent oral hygiene.

Conclusion

Periodontal diseases in healthy children other than chronic gingivitis are uncommon. This should alert clinicians to investigate underlying systemic diseases that the child might have. Routine dental examination in the primary dentition should include a screening of periodontal pockets using a periodontal probe. Bitewing radiographs of the primary dentition should be interpreted thoroughly both for the teeth and periodontal structures. If systemic disease is confirmed, treatment options should involve a survival rate of such systemic disease into consideration. Palliative therapy is preferred to aggressive treatment in terminally ill patients.

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Examining the Child with Periodontal Disease

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Periodontal infections are among the most prevalent of childhood and adolescent diseases in the Western world.[1] Destructive forms of periodontal disease can occur in children and adolescents, and in some cases may be associated with underlying disease. A position paper prepared by The American Academy of Periodontology in 1996, acknowledged four clinically distinct periodontal infections that affect young individuals[2]. These are gingivitis, early onset periodontitis, necrotising ulcerative gingivitis/periodontitis, and periodontitis associated with systemic disease.

Chronic gingivitis is common in children. Past studies in children of different ages have shown that the prevalence and severity of gingivitis increases with age. Early onset periodontitis includes periodontitis that affects young individuals who are otherwise healthy, such as localised and generalised juvenile periodontitis. These can be distinguished clinically and radiographically. Evidence is accumulating that they also differ microbiologically. Necrotising ulcerative gingivitis/periodontitis is more common in children from underdeveloped countries, but may be seen in immunocompromised children in western countries. As in adults, periodontitis associated with systemic disease can occur in children. Some of these include blood dyscrasias (neutropaenia, leukaemia, aplastic anaemia, agranulocytosis, leukocyte adherence deficiency), hypophosphatasia, Chediak-Higashi syndrome, Papillon-Lefevre syndrome, Down syndrome, type I diabetes and HIV infections.

Early diagnosis of periodontal disease is important for successful treatment; it is therefore important that all children receive a periodontal examination as part of their routine dental visits. This paper will review the examination of children with periodontal disease and gingivitis, and will address some of the exciting new technological developments, which have improved methods of detection and diagnosis.

Examining the Gingiva and Periodontium of a Child

The examination should begin with a detailed dental and medical history providing information on past and current medical conditions and medications being taken. Extraorally, the hands and feet should be inspected for abnormalities, such as, hyperkeratosis which together with periodontal disease may be associated with systemic disorders (eg. Papillon Lefevre syndrome). The examination of the mouth should be conducted systematically starting with the oral mucosa, cheeks, lips, tongue, palate and floor of mouth, followed by the examination of the teeth and gingiva, pocket measurements and radiographs. An understanding of the anatomy of a normal periodontium in children is important to comprehend the transition from periodontal health to disease. Gingival health is defined clinically and histologically.[3] Clinically, the gingival tissues are pale pink, firm, and scalloped with knife-edge margins and surface stippling. Histologically, health is characterised by an absence of an inflammatory infiltrate. Clinically, the gingival tissue of young children differs from those of adults; in preschool children the gingivae is more red in colour due to increased vascularity and thinner from less keratinised epithelium[4]. The contour of the marginal gingiva has a "rolled" shape due to the cervical constriction of primary teeth.[4] The sulcular probing depth around primary teeth averages approximately 2mm; buccal and lingual sites are shallower than proximal sites. The alveolar bone surrounding primary teeth shows fewer trabeculae, larger marrow spaces and less calcification, and the periodontal ligament (PDL) is wider than in adults.[4]

The gingival sulcus in children is normally about 0.5mm deep.[5] The junctional epithelium attaches to the enamel via the epithelial attachment, which consists of a basal lamina and hemidesmosomes. The most apical extent of normal healthy junctional epithelium is at the cemento-enamel junction (CEJ) and is only a few cells wide.[5] The most coronal surface of junctional epithelium forms the bottom of the gingival sulcus, where it is approximately 0.15mm wide and comprises 15-30 cell layers.[5] In health, the most coronal level of the connective tissue attachment is at the CEJ. There are numerous collagen fibre bundles in the gingival connective tissues. The fibres of the PDL provide attachment between the root cementum and alveolar bone.[5]

Gingivitis is an inflammatory lesion, mediated by host/parasite interactions; it remains limited to the gingival tissues without involving the underlying PDL, cementum or alveolar bone.[1] The apical extent of the junctional epithelium and coronal attachment level remains at the CEJ.[5] The disease may remain in this state or, with sustained pathogenic bacteria, progress to periodontitis.[3] Clinical signs of gingival inflammation include changes in colour and texture of the marginal tissue and tendency to bleed on gentle probing.[6] This may be accompanied by slight enlargement of the tissues with some loss of firmness and adaptation to the teeth.[5] The tissues often appear red, glossy and oedematous; "bleeding on probingÓ to the bottom of the sulcus is an important indicator of disease as it is associated with an inflammatory cell infiltrate. Various indices such as the Gingival Index (Loe and Silness), have been developed to describe gingivitis in epidemiological and clinical research.[6] The model of experimental gingivitis developed by Loe, Theilade and Jensen in 1965 has been used to investigate gingivitis in children.[7] Longitudinal and cross-sectional studies in children of different ages have shown that the prevalence and severity of gingivitis increases with age. In general, it was found that the younger the child, the less the severity of the gingival changes compared to adults with a similar plaque index.[8] The exact reason for this is not known, but thought to be due to the response of children's gingival tissue to plaque being different from that of adults and changes in the plaque microflora. The initial lesion in childhood gingivitis is composed mainly of untransformed B lymphocytes, in comparison with adults

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where there is a greater density of plasma cells. The microflora increases in complexity with age.
Periodontitis is an inflammatory lesion mediated by host/parasite interactions and results in loss of connective tissue attachment of the root cementum and adjacent alveolar bone, with destruction of the supporting tissues of the periodontium.[1] In teenagers and young adults, localised juvenile periodontitis (LJP) is characterised by severe loss of alveolar bone around permanent teeth frequently localised to the permanent first molars and incisors. In contrast, in generalised juvenile periodontitis (GJP), clinically and radiographically, there is widespread distribution and destruction of the periodontium.[2] A variety of neutrophil functional defects have been reported in patients with LJP and GJP, which may play a role in the clinical course of disease. Periodontitis associated with leukocyte adherence deficiency often begins at the time of primary tooth eruption up to the age of 4-5 years and is termed 'prepubertal periodontitis'.[2] The disease occurs in localised and generalised forms. Since periodontal disease includes inflammatory alterations of the gingiva and a progressive loss of PDL and alveolar bone, the examination must include measures describing such pathological alterations.

Criteria for Assessing Periodontal Disease

To evaluate the amount of tissue lost in periodontal disease and to identify apical extension of the inflammatory lesion, several parameters are used including pocket depth (probing depth), attachment level, furcation involvement, radiographic examination and tooth mobility. Each of these parameters will be discussed in more detail.

Assessment of pocket depth

The pocket depth is the distance from the gingival margin to the bottom of the gingival pocket, measured by a blunt graduated periodontal probe. The pocket depth should be assessed at each surface of all teeth in the dentition and recorded in a periodontal chart. The pocket depth measurements only in rare situations (where the gingival margin coincides with the CEJ) give proper information on the extent of loss of attachment. This can be seen when inflammatory changes in the free gingiva cause swelling and coronal displacement without migration of the dentogingival epithelium to a level apical to the cementoenamel junction (CEJ), resulting in a "pseudopocket".[6]

Assessment of attachment level

This is a measurement using a graduated probe expressed in millimetres from the CEJ to the bottom of the gingival pocket. The advantage of this technique is that the CEJ constitutes a fixed reference point, whereas the gingival margin does not.[5] The use of the Community Periodontal Index of Treatment Needs (CPITN) was adopted by the Federation Dentaire Internationale (FDI, 1985) for use both in epidemiological studies and in screening patients in general dental practice.6 Three indicators of periodontal status are used for this assessment: 1. Presence or absence of gingival bleeding, 2. Supra- or subgingival calculus, 3. Periodontal pockets, subdivided into shallow (4-5mm) or deep (6mm or more).
In the CPITN assessment, a graduated probe with a 0.5mm diameter 'ball point' is used.3 The mouth is divided into sextants and one recording is made for each sextant.[9] In children (under 20 years of age), examination can be restricted to six index teeth, usually the first molars and a maxillary and mandibular central incisor.[3]

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This modification is made in order to avoid classifying the deepened crevices associated with eruption as periodontal pockets.[8] The CPITN codes range from zero to four:- Code 0 - Healthy, Code 1 - Bleeding on probing only is seen, Code 2 - No pockets exceeding 3mm are seen or calculus is present, Code 3 - Pocket depth between 4-5mm, Code 4 - Pocket depth greater than 6mm[8] In 1992, a new system, the Periodontal Screening and Recording (PSR) index was introduced and endorsed by the American Academy of Periodontology.[10] This is a modification of CPITN. Scoring is similar for each sextant of the dentition, with the additional symbol (*) denoting additional periodontal abnormalities, such as furcation involvement, tooth mobility, mucogingival defects and recession.10 The use of the PSR system in early detection of periodontal disease in children and adolescents has been shown to take less time and was better accepted by patients than standard evaluation with a periodontal probe.[11]
A variety of factors can influence the result of measurements made by periodontal probes, such as the thickness of the probe, malposition of the probe due to anatomical features, and the pressure applied on the instrument during probing.6 Controlled-force instruments which provide accurate and consistent measurements of pocket depth and attachment level have been developed.[12] Several are available, one of which is the Florida Probe. This provides a constant probing force, a precise electronic measurement, and computer storage of data.[12] The reproducibility of the Florida Probe when compared to conventional manual probing was found to be significantly superior.[13]

Assessment of furcation involvement and mobility

During the progression of periodontal disease around multirooted teeth, supporting structures of the furcation may be destroyed.[6] Furcation involvements can be classified as: Degree one - horizontal loss of supporting tissues not exceeding one third of the width of the tooth; Degree two - horizontal loss of supporting tissues exceeding one third of the width of the tooth, but not encompassing the total width of the furcation area; and Degree three - horizontal "through and through" destruction of the supporting tissues in the furcation.[6]
The continuous loss of supporting tissue in progressive periodontal disease results in increased tooth mobility. Plaque associated periodontal disease is not the only cause of increased tooth mobility. Overloading of teeth, trauma, and root resorption prior to natural exfoliation may also result in tooth hypermobility.[6] It is therefore important to ascertain the cause of the observed hypermobility.[6] Furcation involvement and mobility of molars in children make mechanical treatment difficult to achieve and may cause discomfort during function.

Radiographic examination

Radiographic evaluation is an integral part of periodontal assessment. The natural history of changes in attachment level in the primary dentition shows the distance from alveolar bone crest to the CEJ seems to increase with age.[14] Radiographic examination is based on each patient's unique needs.[15] Most assessments of progressive alveolar bone loss in clinical practice today is achieved by interpretation, ie, visual comparison of radiographs taken over time. The height of the alveolar bone and outline of the bone crest are examined radiographically,[6] and can be compared with the CEJ.[4] Unfortunately, it is difficult to detect small changes in bone that occur between examinations using cortical and trabecular bone since conventional bitewings or periapical radiographs do not usually detect bone loss until 30% of the bone mass is destroyed.[16]
Digital radiography was introduced to dentistry in the 1980's.[15] Using digital subtraction radiography and standardised radiographs,

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small changes in bone status can be identified.[16] This technique is used to detect small changes in hard tissues that occur between examinations. In brief, digital subtraction radiography uses specialised computer programs to remove all structures that have not changed from a set of two radiograph films taken at different examinations.[15] This can help the clinician assess the rate of alveolar bone destruction in children before clinical signs of increased mobility are detected, allowing early treatment to commence, and monitoring of the progress of periodontal disease.

Systemic considerations

A general medical evaluation is needed to determine if systemic diseases are present for children exhibiting severe periodontitis, especially if the disease appears resistant to therapy. Laboratory tests should include: a full blood examination, white cell count and differential to detect blood dyscrasias, serum alkaline phosphatase and urinary phosphoethanolamine (the former is decreased and the latter increased in hypophosphatasia), and blood glucose levels (random and fasting) to screen for diabetes mellitus. Tissue biopsy may be required if Langerhan's Cell Histiocytosis is suspected.

New Tests for Periodontal Disease

Traditional methods used for determining the progression of periodontitis assess the degree of damage that has occurred to the periodontal tissue over time. Supplementary diagnostic tests can be used to perform two basic tasks.[15] The first is screening, ie, to separate patients with and without disease. The second is to detect high risk, progressive disease sites in patients. The clinical value of fully validated diagnostic tests is considerable because they are potentially useful in identifying the presence of putative pathogens, monitoring the response to therapy, identifying sites at high risk for progression, and assisting the clinician in determining a patient specific recall for supportive periodontal therapy.[15]

A large number of supplemental diagnostic tests are currently available. Supplemental diagnostic tests can be used to detect the presence of bacterial pathogens, host-derived enzymes, tissue-breakdown products and inflammatory mediators.

Detection of putative bacterial pathogens

Several strategies have been developed to detect putative periodontopathogens, such as *Bacteroides species (Bacteroides forsthus, Porphyromonas gingivalis, Prevotella intermedia), Actinobacillus actinomycetemcomitans, Spirochetes (Treponema denticola)*. They include culture, microscopic examination, DNA (or RNA) analyses, detection of specific bacterial antigens, and detection of specific bacterial enzymes. Bacterial culturing relies on the presence of viable micro-organisms and are likely to show variations from sample to sample due to technical limitations.[15] It is also expensive and time consuming, but does allow bacterial antibiotic sensitivity to be determined. Such analyses of the flora can only distinguish between motile and nonmotile bacteria and categorise them into general morphotypes. Determination of genus, species and antibiotic sensitivities is not possible.[15] Indirect bacterial detection methods such as DNA probes and enzyme linked immunological assays do not require the maintenance of organism viability after sampling and have been used to detect subgingival periodontal pathogens.[17]

Traditional methods used for determining the progression of periodontitis assess the degree of damage that has occurred to the periodontal tissue over time.

Nucleic acid probes

The bacterial DNA encodes information for protein synthesis and gives individual bacteria their specificity. Individual strains of the same bacterial species can differ in nucleotide sequence and can therefore be distinguished by their DNA.[18] In recent years, DNA probes have been developed to identify nucleotide sequences that are specific for bacteria believed to be of diagnostic significance, including suspected periodontal pathogens.[19]

Bacterial DNA recovered from the subgingival sulcus is first enzymatically denatured (using multiple restriction endonucleases), to single strands and combined on filter paper with radio-labelled single strands of DNA (DNA probe) from a known bacterium.[15] If the plaque specimen contains the known microbe, its DNA will hybridise with the DNA probe and can be identified as a dark spot on the radiograph film due to the radioactive decay of phosphorous (P^{32}).[17] The intensity of the spot varies with the number of organisms present in the specimen and therefore provides quantitation.

Probes using RNA can be constructed using the nucleotide sequence for ribosomal RNA (rRNA) which is essential for protein synthesis.[18] Commercial DNA/RNA probes such as 'Omnigene' (Omnigene Inc., Dental Diagnostics, Cambridge, MA) are available, and are sensitive enough to identify as few as 1000 bacterial cells per plaque sample.[17]

Detection of antigens of suspected pathogens

The use of immunological techniques such as immunofluorescence or enzyme linked immunosorbent assay (ELISA) can detect individual bacterial species.[20] These techniques use specific antibodies (monoclonal or polyclonal) which bind to the selected bacterial antigens and are then detected by labelling the primary antibody directly with a fluorescent marker (direct immunofluorescence) or with a fluorescent secondary antibody (indirect immunofluorescence).[21]

In the ELISA assay the primary antibody is detected through a colourimetric reaction which is catalysed through an enzyme, usually horseradish peroxidase or alkaline phosphatase linked to an antibody.[3] This assay is very sensitive and relatively economical to run.[3] A rapid chairside assay, "Evalusite" (Kodak Dental Products, Hemel Hempstead, UK) is based on a filter-separation enzyme immunoassay which detects antigens on *P. gingivalis, P. intermedia and A. actinomycetecomitans* to a level of 10^4 bacterial cells per sample.[22] The assay takes approximately two minutes to provide the result from a paper point sample of the subgingival microflora.[22]

Latex agglutination is an immunologic assay with moderate sensitivity that is based on the binding of protein to latex and its agglutination or dumping on exposure to its specific antigen or antibody.[3] It has been used to detect antigens of *P.gingivalis, A. actinomycetecomitans,* and *P. intermedia* samples.[23]

Detection of enzymatic activity of suspected pathogens

Some periodontal pathogens produce a trypsin-like enzyme capable of hydrolysing beta-naphthylamide derivatives, mainly N-alpha-benzoyl-DL-arginine-2-naphthylamide (BANA). This protease is produced by *Porphyromonas gingivalis* and to a lesser extent *Bacteroides forsythus* and *Treponema denticola.*[19] BANA is colourless, but on hydrolysis beta-naphthylamide is released and reacts with a variety of dyes to form coloured products.[15] A rapid chairside BANA hydrolysis test, 'Perioscan' (Oral B, Aylesbury, UK) has been developed that can identify plaques containing one or more of these bacteria.[24] Several cross sectional studies have employed this simple test and have reported BANA-positive sites in areas of destruction

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and BANA-negative sites in healthy sites.[24] The main draw backs are a lack of quantitative data and the inability to determine which of the three bacteria are responsible for the enzyme production.

Detection of host derived enzymes, mediators and breakdown products

Much attention has been directed to the measurement of local levels of specific byproducts of the periodontal disease in the gingival crevicular fluid (GCF) such as enzymes, bacteria-specific immunoglobulins, soluble inflammatory mediators including cytokines, and cellular or tissue degradation products.[15] Some of these substances have been suggested as possible markers for the detection of active periodontal lesions.[15]

Among the host-derived enzymes present in GCF that have received much attention are aspartate aminotransferase (AST), collagenase, and related neutral proteases, beta glucoronidase and elastase.[15]

The 'Perioguard' kit (Colgate-Palmolive, Guilford, UK) is an assay for levels of AST at selected periodontal sites and may be used at the chairside. The assay is based on the colourimetric detection of oxaloacetate produced by the action of AST on L-aspartate and 2-oxoglutarate.[22] The Perioguard kit was studied recently in a multicentre trial, and when compared with other standard diagnostic procedures, provided improved capacity to distinguish between diseased and non-diseased periodontal sites, allowing better assessment and monitoring of disease.[25] Another chairside kit, 'Periocheck' (AC Tech, Schein Rexodent, Southall) is used to analyse GCF for the presence of proteolytic enzymes released from neutrophils. The test attempts to provide an objective assessment of the inflammatory condition of the periodontium. [22]

A variety of inflammatory mediators are produced by tissues with gingivitis or periodontitis.[15] At present, there are no fully developed and validated diagnostic or prognostic tests for progressive periodontitis that assess the level of inflammatory mediators in GCF.[15] However, preliminary work has suggested that prostagandin E2, interleukin 1 and tumour necrosis factor alpha may be associated with sites affected with periodontitis.[15,26] Gingival crevicular fluid based tests appear promising, however more work needs to be done before they have been fully tested and validated.[15] The GCF levels of hydroxyproline (from collagen degradation) and glycosaminoglycans (from degradation of ground substance) have been investigated in cross sectional studies.[15] However, despite promising research, no tests are commercially available for these at present.

Future Developments

The Perioscan kit was available in Australia in the early 1990's; since then it has been removed from the market. Many practitioners considered the test was sensitive in identifying "disease sites", but it had a low level of specificity giving false positives to many healthy sites. The Periocheck and Perioguard are currently not available in Australia. Current literature suggests that Perioguard appears to be a promising test for the future. To date there have not been any data on the use of these kits in children. With the continued development of new tests, the diagnosis of periodontal disease has entered a new era. New chairside tests may allow clinicians to assess the potential for disease development or recurrence without waiting for clinical signs of disease. This becomes increasingly important in children with early onset periodontitis.

The new chairside tests must be able to provide additional information which cannot be gained from traditional methods of pocket probing and radiographic examination. This means that sites of potential disease risk should be detected rather than sites where disease has already occurred, allowing diagnosis and treatment to be made earlier, ultimately improving the prognosis of periodontal disease in children.

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Abstracts from the Colgate Oral Care Paediatric Dentistry Graduate Student Award

Tooth Wear in the Deciduous Dentition of South Australian Children

Dr. Susan Springbett

Most studies of the nature and extent of tooth wear in human populations, including the processes of abrasion, attrition, erosion and fracture, have concentrated on the permanent dentition. However, recent studies have indicated that erosion is likely to be a major cause of tooth wear in the deciduous dentition, possibly linked with dietary patterns.

The aim of this study was to determine which of the wear processes predominate in the deciduous dentition of South Australian children and to assess whether any relationship exists between the extent of wear observed and dietary intake or habits. The study forms part of a larger investigation of the extent and rate of deciduous tooth wear observed on dental models of children from different populations. Thirty-six children (22 boys and 14 girls) aged between 9.4 and 11.3 years (mean 10.6 years) were included in the present study. Information obtained for each child included a clinical examination, dental models, saliva pH recordings, a questionnaire about habits and medical history and a three-day dietary sheet. A scoring system was developed to determine the type and extent of wear observed on deciduous canines and molars and an overall score assigned to each tooth using a modified form of the Smith and Knight Tooth Wear Index. The dietary sheets were scored using a scoring system that was developed based on work done by Jarvinen and colleagues.

It was found that 85 percent of deciduous teeth had dentine exposed, the extent varying from small pits to complete loss of enamel. The deciduous canines displayed highest scores for exposed dentine. Eighty-nine percent of teeth displayed wear facets that had either distinct or indistinct borders. The crowns of all deciduous maxillary canines showed evidence of faceting. Chipping or fracture of enamel was most evident on the deciduous molars (up to 18 percent).

The prevalence of tooth wear was similar in boys and girls and there were no significant differences between teeth on right or left sides. There was some evidence that the maxillary teeth displayed greater wear that the corresponding mandibular teeth. Seventy-five percent of individuals had dietary scores that indicated moderate to high acid intake. Unstimulated saliva pH recordings between 6 and 7 were obtained for 50 percent (18) of the sample.

In summary, it is apparent that there is considerable wear present on the deciduous teeth of the children in this study. The wear processes of attrition and erosion are most common, with few individuals also displaying enamel fracture. The dietary records of the children confirm a high lever of acidic dietary intake. The potential for a combination of low unstimulated saliva pH and an acidic diet to contributed to extensive tooth wear in the deciduous dentition is an area that deserves further investigation.

A Study of the Dental Treatment Needs of Children with Disabilities in Victoria

Dr. Mala Desai

Dental disease is a major health problem for persons with disabilities. A high prevalence of poor oral hygiene, periodontal disease, and malocclusion among the population with disabilities has been cited frequently in the literature. The dental needs of children and adolescents in Victoria have received little attention over the past decade, and information on prevalence or oral disease in various disability groups is sparse. The objectives of this study were to: (1) record the prevalence and severity of oral disease of a selected age group (9 to 13 years) of children with disabilities in Victoria; (2) survey the treatment needs of the study population with reference to dental caries, periodontal disease and malocclusion; (3) investigate associations between dental health status and treatment needs; and (4) determine what interventions are most appropriate with reference to the policies and planning of Dental Health Services Victoria. Three hundred children (150 attending special developmental schools (SDS) and 150 attending special schools (SS), ages 9 to 13 years (mean age, 11 years) participated in the study. Oral examinations were conducted and data was recorded on a form. Questionnaires were completed by parents or guardians to provide biographic data and information on the child's medical history, current medications, perceived dental problems, nature of disability, and habits and level of function. The dependent variables were: the dental status and treatment needs, and overall treatment need recommendations for each subject. There was one independent variable, the level of function, which was a qualitative summation of the levels of dependence of the subject over five self-care activities (brushing teeth, feeding self, dressing self, walking alone, and going to the toilet alone). Subjects were grouped according to levels of function and comparisons made between the six sub-groups. The caries experience of children in the SS was lower than in the SDS (d+D 1.3$\pm$1.6 vs 1.5 $\pm$2.4; dmft+DMFT: 2.0$\pm$2.3 vs 2.5$\pm$3.1). There were significant associations between the number of decayed teeth and the dmft+DMFT index and level of function of subjects ($p<0.005$). A higher prevalence of developmental defects of the enamel in SS children (2.2$\pm$4.1) compared with SDS children (1.2$\pm$2.8) was recorded. Children attending SDS had higher unmet preventive and/or restorative treatment needs compared with SS children. The prevalence of periodontal disease was very high, representing 90% of the study sample. There was a significant association between level of function and periodontal status ($p<0.005$), and need for periodontal therapy ($p<0.005$). The major treatment requirement for periodontal conditions was scaling and cleaning together with oral hygiene advice. Malocclusion was present in 92% of subjects and ranged in severity from slight occlusal irregularities to gross dentofacial anomalies, hence indicating a great need for orthodontic treatment. With reference to level of function , the majority of children (70%) required assistance with brushing their teeth. Children who were more dependent on care-givers for self-care activities, tended to have higher levels of dental disease, and consequently higher treatment needs. Recommendations were made to improve the dental health status of children with disabilities; these included the implementation and/or expansion of dental services, oral health promotion and preventive services, professional education, and research and evaluation.

Three Dimensional Reconstruction of the Medial Edge Epithelial Seam during Palatal Fusion.

Dr. Suzanne Brent

A study was undertaken to analyse the breakdown of the medial edge epithelial seam that occurs during normal palatal fusion by reconstructing the disintegrating midline seam

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in epoxy resin. The specimens were orientated to permit sectioning in the sagittal plane and the block trimmed to the region of the epithelial seam. Sections of the full length of the palate were taken at 1 micron intervals without loss from the point of first appearance of the epithelium until sectioning had passed completely through the line of fusion. Sections were stained with toludine blue and sequential light microscope images digitised and processed using the three dimensional analysis software packages, Voxblast and Voxelview. A three dimensional image of the epithelium seam across the line of fusion was this reconstructed. A total of 30 embryos were sectioned and 7 were reconstructed. At the light microscope level the sagittal view revealed true islands of epithelium in only one specimen. Most frequently chords of cells were visible, wither completely crossing the thickness of the palate and uniting the nasal and oral surfaces, or projecting like fingers up from the oral surface. Ultrastructural evidence supported the involvement of apoptosis in the removal of the medial edge epithelium. The three dimensional reconstructions provided a unique representation of the nature of disintegrating medial edge epithelial seam. Initiation of breakdown was observed most frequently toward the nasal side of the seam. However, the earliest single complete breaches, were located in the middle of the palatal shelf.

Immunohistochemical Analysis of Morphogens and Death Proteins in Palatal Fusion

Chinh Nguyen, Angus Cameron, John Gibbins, and Neil Hunter.

Palatal fusion is an important process in oro-facial development, yet it is poorly understand despite of having an incidence of about 1:1000 live births. The formation of the secondary palate by fusion of the apposing shelves is critically mediated by the formation and subsequent dispersion of the mid-line epithelial seam. Three theories have been proposed to account for the dispersal of the epithelial seam to allow mesenchymal confluence. These are: (1) programmed cell death (PCD or Apoptosis), (2) epithelial-mesenchymal transformation (EMT), and (3) migration of cells from the epithelial seam to nasal and oral surfaces.

Morphogenic proteins of the transforming growth factor-beta (TGF-B) family have the potential to regulate all three aspects of epithelial seam response. On this basis, utilising a foetal rate model, specific antibodies were used to probe for the presence of the morphogens and their receptors. Also studied were BCL-2 and BAX, proteins reported to influence cellular susceptibility to death. A coordinated expression of TGF-B 1,2 and 3 was observed in relation to the fusion process. Strong staining in residual islands of the mid-line epithelial cells preceded lateral spread in the oral and nasal epithelial aspects of the palatal shelves. Similar findings were obtained for the protective anti-death protein BCL-2 and its antagonist BAX. Strong expression of these proteins was concentrated in the epithelial seam in the presence of dead seam cells. The data was compatible with an important role for the morphogens in palatal fusion. Expression of BCL-2 and BAX appeared to correlate with the sequence rather than with death of specific cells.

Morphogenic proteins of the transforming growth factor-beta (TGF-B) family have the potential to regulate all three aspects of epithelial seam response.

A Comparative Study of the Cariogenicity of Infant Formula and Bovine Milk in Rats

Dr. John M. Sheahan

There is a paucity of knowledge regarding the cariogenicity of infant formulae, its role in nursing caries, and the ability of mutans streptococci to colonise the oral environment when the only available fermentable substrates are those present in either bovine milk (BM) or a whey dominant infant formula (IF). Therefore, the objectives of the present study were: to compare the cariogenicity of a whey dominant IF and the cariogenicity of BM in rats that are fed each substrate ad libitum, and to investigate the ability of Streptococcus mutans Ingbritt to infect rat dental fissures when the only available fermentable substrates are those present i either BM or a whey dominant IF. Forty-five Sprague Dawley rats (pups of specific pathogen free, barrier-sustained dams) were randomly divided into 3 groups, weaned and then infected with streptomycin resistant Streptococcus mutans Ingbritt (SRSMI) when they were 18, 19, 20 and 21 days old. The SRSMI had been grown on tryptone yeast salts lactose agar. After weaning, BM, IF or infant formula plus 20 per cent sucrose (IF+S) was fed to the rats. During this period the provided diet formed the only source of nutrition. Saliva was collected from the rats and they were terminated six weeks after weaning. Molar fissure plaque was sampled and the presence of SRSMI was subsequently confirmed in all rats. No significant differences were detected between the groups in the number of micro-organisms recovered. Caries was assessed after murexide staining. There was almost complete absence of caries in the BM group. Significant differences in the number of buccal and lingual carious lesions (BM 0.0, IF 0.3, IF+S 1.0), morsal carious lesions (BM 0.1, IF 2.6, IF+S 3.2) and sulcal carious lesions (BM 0.0, IF 3.9, IF+S 5.5) were detected between the BM group and each of the other two groups. Significant differences in the extent of the carious lesions were also detected between the BM group and each of the other two groups. For example, the scores at the moderate dentinal (Dm) level were: BM 0.0, IF 0.9, IF+S 1.2 for morsal carious lesions; and BM 0.0, IF 4.3, IF+S 8.1 for sulcal carious lesions. There were significant differences in the weights and lengths of the rats in the three groups at termination due to the caloric differences in the diets and the diets' organoleptic properties. The difference in caries activity was not attributed to a difference in salivary function as there was no significant difference in the salivary volume or salivary protein, calcium and phosphate concentrations between the groups. This suggests that an adequate amount of protein, calcium and phosphate was derived from the IF. The significant differences in caries are consistent with the lower concentrations of the caries protective calcium, inorganic phosphate and casein, and the higher concentration of lactose in IF compared with BM.

Trauma to Anterior Teeth Intruded Maxillary Permanent Cental Incisor - A Case Report

Dr. Soni Stephen / Dr. Paul Marin

Trauma to anterior teeth is a common injury seen in children. Intrusion of permanent anterior teeth is a severe traumatic injury. The onset of an inflammatory root resorption can be a problem for the prognosis of the tooth involved and often the treatment has to be modified to deal with the root resorption. This paper reports

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the successful management of a case of intruded upper permanent anterior teeth with inflammatory root resorption. The benefits of using Ledermix as a dressing is discussed and various treatment phases are recorded on radiographs and photographs. This successful case demonstrates the effectiveness of timely intervention and use of Ledermix in such situations.

The Demand for Dental General Anaesthesia in Children at Westmead Hospital, Sydney

E. Alcaino and N.M. Kilpatrick

The use of general anaesthesia (GA) to provide dental treatment for children has been reported for several decades. The aim of this study was to assess demographic factors and nature of treatment provided for children treated under GA at Westmead Hospital. Randomized samples of patients from 1984 and 1996 were selected and data collated in a retrospective manner; information such as age, suburb or residence, waiting list time, reason and source of referral, ethnicity and treatment provided were recorded. The data was analysed using T-tests and Chi-square tests. Results showed that certain populations of Sydney provide greater number of children requiring GA services. There was a steady increase in the demand for this service, from a total of 212 children in 1984 to 777 in 1996. Dental caries was the main reason for referral, with an increase from 70% in 1984 to 83% in 1996 (p,0.05), with over two thirds of the children being under 6 years of age. the ethnicity of these children has changed over the 12 year period; with significantly more Asian and Middle Eastern children being treated (p,0.05). General practitioner dentists, self-emergency patients and The School Dental Service were the main sources of referrals. The nature of treatment has changed significantly with 20% more primary teeth being extracted in 1996 than in 1984. The mean waiting time increased from 37 to 80 days in the 12 year period, indicating that the demand has outstripped its provision. The increase in demand for GA services may be attributed to: changes in pattern of caries distribution, changes in demographics, increased restrictions for GA services in the private sector, and changes in the public dental services. *Despite a general decline in caries in Australia, nursing and rampant decay still remain a significant problem in certain communities within Sydney. The demand for dental treatment under general anaesthesia has increased over the past two decades with caries remaining the main reason for use of this service.*

Child Abuse...from page 1
ing a suspected case. Most respondents (74%) knew that they could be called in front of the Children's Court to give evidence. However, respondents were not well informed on a number of major aspects of the problem. Few respondents included neglect (20%) or sexual abuse (33%) in their definition, and only 41% considered oro-dental findings amongst the signs of the condition. Few respondents (29%) were aware that current reports indicate that child abuse is not more prevalent in particular socioeconomic groups and that the scope of the problem knows no social, educational or financial boundaries. Less that half the respondents (49%) could name appropriate avenues for reporting, 44% knew that the identity of a reporting dentist remains confidential at the initial report, and only 24% were aware that they are not legally required to report suspected cases in Victoria. This lack of knowledge was also demonstrated by the high proportion of respondents who chose not to provide responses to questions concerning their management of a suspected case, and who chose the "don't know" option on several multiple choice questions.

Between-group comparisons showed that members of ANZSPD were more knowledgeable in some aspects of the problem than those in the ADA, NG and ASE groups. This was attributed in part to their relatively greater personal experience of the problem (54% ANZSPD members had suspected a case and 23% had reported a case, vs 20-27% suspicion and 3-16% reports for ADA, NG and ASE groups), and to the attendance of some members at a half day seminar on child abuse held in 1995 by ANZSPD. Respondents in all four groups demonstrated similar attitudes towards reporting a suspected case of child abuse, indicating that certainty of diagnosis (82-89% affirmative responses) and possible effects on the child (76-87%) would be paramount considerations for them, with possible effects on their practices (9-11%) and fear of litigation (16-21%) being minor considerations. Uncertainty of diagnosis by dentists has been demonstrated by other authors as the most common reason for not reporting suspected cases.

Most recently, application of the legislation governing mandatory reporting of child abuse in Victoria has been tested. On December 5, 1997, the Ringwood Magistrates Court dismissed the first Victorian case of a mandated professional charged with failing to report suspected child abuse. In this case, a school principal was charged with failing to report that a five year old student had claimed his father sexually assaulted him. The principal was acquitted because it could not be proven beyond reasonable doubt that she had formed the "belief" on reasonable grounds that the child had been abused. While the court decision raises doubts about the effectiveness of the legislation, the primary issue for both mandated and non-mandated professionals is adequate training in the recognition of the various forms of child abuse to ensure their compliance with the legislation, regardless of how it is interpreted. In view of the high likelihood of oro-dental injuries occurring in association with child abuse, and the low reporting of cases by the dental profession which has been reported elsewhere in the literature, this study has demonstrated a clear need for dentists to receive formal training at the levels of undergraduate, postgraduate and continuing professional education in the recognition and reporting of child abuse. The dental profession must become actively involved in the recognition of all types of child abuse. Although not currently mandated in all states of Australia to report the problem, all dentists should address their professional obligation to do so when confronted with a suspected case of child abuse, and should become fully aware of the appropriate reporting procedures in their location.

Acknowledgements

This project was supported by a research grant from the Australian and New Zealand Society of Paediatric Dentistry (Federal office and Victorian Branch); the support of this Society and the participation of its Victorian members in the survey are acknowledged with gratitude. Members of the Australian Dental Association Victorian Branch, the New Graduates group (ADAVB), and the Australian Society of Endodontology Victorian Branch are also thanked for their enthusiasm and willing participation in the survey.

Submitted by Louise Brearley Messer
The Elsdon Storey Professor of
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School of Dental Science
The University of Melbourne
January 30, 1998

Dental Care for Children in the U K

An Undergraduate's Experience

Carmel Trudgeon
(5th Year Dentistry Student, University Of Sydney)

During the 1997-98 Christmas vacation, I left the Australian summer behind and embarked on an exciting journey to "wintry" UK and Europe. I spent a week in Newcastle upon Tyne Dental School (3 hours by train north of London) and one week at Cardiff Dental School in Wales.

The emphasis of the trip was to investigate the provision of dental care for children in the UK (and compare child dental heath in the UK to Australia).
My experiences in the UK were primarily spent in the Community Dental Services. This included:

- observing treatment of children and special needs patients in Community Clinics.
- mobile dental van/caravan visits to schools.
- observing the "screening" protocol for school children.
- home visits to the elderly and housebound.
- discussions with numerous health professionals and parents (of child patients) about "the UK health system and what it provides for children".

The following is an account of what I learnt in regards to dental care for school children in the UK.
The UK's National Health Service (NHS) is divided into 3 broad groups:

1. The General or Family Practitioner Services - provides primary medical, dental, pharmaceutical and optical care for the population through independent practitioners.

2. The Community Services - provides primary care to selected priority groups, including in particular children, elderly and special needs.

3. The Hospital Services - provides specialist care for patients referred from the primary care services.

Over 80% of dentists work in the General Dental Services, a little over 10% work in the Community Dental Services, whilst less than 10% work in the Hospital Services.

General Dental Services: (GDS)

Before October 1990, the GDS was based on a fee-for-item of services payment system for both children and adults. The dentist had a responsibility to render a patient "dentally fit" at the end of a course of treatment, but had no contractual responsibility for providing continuing care. In October 1990, a patient registration scheme was introduced. Therefore, patients who attended a dentist for more than emergency treatment could enter into a long term contractual relationship with their GDP.

Over 80% of dentists work in the General Dental Services, a little over 10% work in the Community Dental Services, whilst less than 10% work in the Hospital Services.

In conjunction with the registration scheme came a capitation scheme for the treatment of children, whereby dentists would receive a fee for each child based on age of the child registered, which was independent of the amount of treatment provided. The aim of this new method of payment was to encourage the prevention of disease rather than its repair and to encourage continuing responsibility rather than an episodic course of treatment.
The bulk of the cost of the GDS is borne by the state. However, it has been government policy since 1951 to impose charges on those patients receiving treatment who can afford it. Currently, all dental treatment for children under 18, students under 19, pregnant women, nursing mothers and those in receipt of Income Support or Family Credit is free. Since 1988, all other patients pay 80% of the cost of each course of treatment up to a maximum charge of 350.

Community Dental Services: (CDS)

(the predominant service I experienced during the trip).
In 1989 the Department of Health identified specific roles for the CDS:

1. to monitor levels of dental health through the population.
2. to provide a school based screening programme.
3. to provide dental health education and preventative programmes.
4. to co-ordinate oral health promotion strategies.
5. to provide a full range of treatment to patients who are unable or unwilling to obtain treatment from the general dental services.

Thus, the CDS is essentially a safety net treatment service.

The CDS also provides:

- orthodontic treatment
- domiciliary care (home visits for the elderly)
- emergency care (on weekends and after hours)
- care for phobic patients
- care for physically and mentally disabled (good wheelchair access to most clinics)
- mobile clinics (for special needs schools and schools with high treatment needs e.g. low socio economic areas where access to a dentist is difficult and uptake of dental services is low).

There are no charges incurred by the patient under the CDS, except for dentures and

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bridges.
General dentists often refer to the CDS for the following reasons:

- a non compliant/difficult child patient - more time is required in a more relaxed atmosphere.
- for nervous patients - RA is available in CDS.

The UK is divided into many Local Health Authorities an each uses their allocated funds to run their CDS independently.
Topics of particular interest during my UK trip included:

1. Water Fluoridation vs Caries Rate:

In the UK, fluoride has been the subject of much controversy as to whether it is of benefit and safe. Water fluoridation is limited to the West Midlands and Newcastle upon Tyne. I noticed a dramatic difference in the prevalence of rampant caries in children in Cardiff (non-fluoridated) as opposed to Newcastle (fluoridated) (Table 1). On a mobile dental van visit in Cardiff, children presented with abscesses and gross carious lesions. Extractions were a common treatment option. In Newcastle, caries was less prevalent possibly due to water fluoridation. Treatment tended to have a preventative focus e.g fissure sealants and conservative restorations.

2. Water Fluoridation and Socio-economic status

Research has shown that, generally speaking children from poorer backgrounds are the most prone to tooth decay. This is because they and their families are less likely to eat a sugar free or low sugar diet, less likely to be able to afford fluoride toothpaste and less likely to be registered with a general dentist who can give advice on preventative measures and spot early signs of potential problems.
Studies caries out in Newcastle (fluoridated) and Northumberland (non-fluoridated) on 5 year old children have shown that children in the lower social class groups benefit the most from fluoridation. Thus, water fluoridation is a mass means of closing the gap on dental inequality.
Despite the benefits of water fluoridation, the present legislation in the UK is that the private Water authorities/companies make the final decision on whether or not they fluoridate the water, even if the heath authority has advised them to do so. Thus, the water fluoridation issue continues to be an endless battle for profluoride groups.

3. School Screening Programmes:

The aim of dental screening is to encourage the uptake of dental care by all children, especially those in whom disease is recognised.
In the UK, the CDS in each Local Health Authority designs and implements their own school screening programmes.
I have experienced the screening systems of:
(a) Northumberland (a local authority of Newcastle upon Tyne).
(b) South Glamorgan (a local authority of Wales)
(c) NSW, Australia (S.O.K.S - Save Our Kids Smiles)
The major differences which exist include:

- In Australia, screening is usually carried out by dental therapists whereas, in the UK screening must be performed by dentists.
- SOKS visits every government/Catholic school in NSW every year and examines years K, 2, 4, 6 and 8. In the UK, both systems categorise the schools into groups according to perceived treatment need (as demonstrated by previous disease rates) and visit schools at appropriate intervals e.g bi-annually, annually etc.
- Different criteria for assessing disease and hence providing referral letters for treatment exist between the 3 systems.
- In NSW, Australia, the screening procedure is performed with the child in a supine position, with overhead lighting, plus probe and mirror. In the UK, the child stands in front of the dentist and a pencil light and mirror are used for the screening examination.
- SOKS incorporates dental health education into the screening visit e.g. videos, talks, hands-on. In the UK, it is encouraged that the classroom teacher includes Dental Health in the curriculum e.g. teach it as a Maths or Science lesson.

4. Oral Health Promotion:

It is the role of the CDS to devise strategies for improving the dental health of the population. Oral Health Promotion is more than just the responsibility of Dental Health Professionals. It must involve:

- politicians and administrators (e.g. to implement water fluoridation).
- food manufacturers(e.g. to increase the cost of confectionary, to label cariogenic products, to reduce the amount of sugar in food/drink.)
- pharmacists/doctors (e.g. to prescribe and supply sugar free medicines/medicaments).
- teachers, post-natal nurses (e.g. must be knowledgeable about dental issues).

In North Tynside CDS, an oral health promotion strategy with objectives, targets and methodologies has been outlined for different age groups (e.g. children < 5years, children 5 - 18 years, adults, elderly). For example, one objective for the children 5 - 18 years is, promoting the sale of non cariogenic products in school tuck shops.
From my experiences, I can conclude that the dental services in both Australia and the UK have one common aim; To provide the opportunity for everyone to retain a healthy functional dentition for life, by the efficient use and distribution of treatment resources. Dental care for children appears to be well catered for in both the UK and Australia, even though there is a small group of children where the majority of decay is still experienced.
Finally, my trip was far from "all work and no play". I spent many evenings in the local pubs with new found friends. I also had time to visit a few of the castles in the picturesque English countryside. All in all the trip was an invaluable experience. In addition to struggling with the Welsh accent, experiencing below zero temperatures and coping with the short daytime hours (9a.m. to 4.30p.m.), I learnt a lot about UK dentistry. All in all the trip was an invaluable experience.
I would like to thank the NSW Branch of the Australian and New Zealand Society of Paediatric Dentistry and Oral B for awarding me this travel scholarship. Also many thanks to Dr Kilpatrick (Westmead Hospital), Dr Nunn (Newcastle) and Dr Treasure (Cardiff) for their time and organisation. I hope that this scholarship will continue to help others like myself have the opportunity to travel and broaden their horizons.

Table 1

		DMFT (5yr olds) 1995	% Population Fluoridated
Newcastle upon Tyne	North Tyneside	1.1	50
	Newcastle	1.5	100
	Northumberland	2.0	35
Wales	South Glamorgan	1.9	0
	Mid Glamorgan	2.8	0
	West Glamorgan	2.9	0
Australia		1.4 (1987-88)	>80

Congratulations to Dr Suzanne Brent

Dr Brent won the Colgate Oral Care Paediatric Dentistry Graduate Student Award at the 11th Biennial Meeting of the Australian and New Zealand Society of Paediatric Dentistry in Sydney last August. She has recently returned from the European Academy of Paediatric Dentistry Conference in Sardinia having also won the research prize there. The prize was awarded by the Italian Academy of Paediatric Dentistry for the best poster presentation. She has won $1000 to be spent on "An educational experience in Italy!" Dr Brent has now completed her Masters Degree in Paediatric Dentistry and is currently working for the university (Sydney) whilst planning to set up a private specialist practice.

Dr Nicky Kilpatrick

Coming Events

World Congress on Preventive Dentistry

Capetown, South Africa
8-11 October 1998.
Contact: The Secretariat, WCPD 1998, PO Box 95031,
Waterkloof, Pretoria 0145, South Africa

86th FDI World Dental Congress

Barcelona, Spain
8-12 October 1998.
Contact: FDI World Dental Federation,
Congress Department,
7 Carlisle Street,
London W1V 5RG, UK

10th International Conference on Dental Traumatology

Melbourne, Australia
19-21 March 1999.
Contact: PR Conference Consultants,
113 Whitehorse Road,
Balwyn Victoria 3103
Australia

17th Congress of the International Association of Paediatric Dentistry

London, UK
2-4 September 1999.
Contact: Concorde Services Ltd,
10 Wendell Road,
London W12 9RT UK

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